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The microbicidal agent C31G inhibits Chlamydia trachomatis infectivity in vitro.

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Safe and effective vaginal microbicidal compounds are being sought to offer women an independent method for protection against transmission of sexually acquired pathogens. The purpose of this study was to examine the efficacy of two formulations of one such compound, C31G, against Chlamydia trachomatis serovar E alone, its host epithelial cell (HEC-1B) alone, and against chlamydiae-infected HEC-1B cells. Preexposure of isolated, purified infectious chlamydial elementary bodies (EB) to C31G, at pHs 7.2 and 5.7, for 1 h at 4 degrees C resulted in reduced infectivity of EB for HEC-1B cells. Examination of the C31G-exposed 35S-EB on sodium dodecyl sulfate-polyacrylamide gel electrophoresis autoradiographs and by Western blotting revealed a C31G concentration-dependent and pH-dependent destabilization of the chlamydial envelope, resulting in the release of chlamydial lipopolysaccharide and proteins. Interestingly, when the host human genital columnar epithelial cells were infected with chlamydiae and then exposed to dilute concentrations of C31G which did not alter epithelial cell viability, chlamydial infectivity was also markedly reduced. C31G gained access to the developing chlamydial inclusion causing damage to or destruction of metabolically active reticulate bodies as well as apparent alteration of the inclusion membrane, which resulted in premature escape of chlamydial antigen to the infected epithelial surface. These studies show that the broad-spectrum antiviral and antibacterial microbicide C31G also has antichlamydial activity.

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